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PIERCE'S DISEASE OF THE GRAPEVINE

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Pierce's disease of the grapevine (Vitis spp.) is the principal limiting factor in the culture of grapes in the southeastern United States (8). Both European type (Vitis vinifera L.) and the American type (V. labrusca L.) bunch grapes succumb to the disease. The probable home of the causal agent of Pierce's disease is the southeastern United States, since in this area native Vitis spp. are resistant, and Pierce's disease is endemic. The disease is not a problem in the grape-growing areas of northeastern United States where cold temperatures are not conducive to the survival of the pathogen (16). In California, Pierce's disease has destroyed some 75,000 acres of grapevines in epidemics that have occurred since the first major epidemic in southern California in the late 1880s (8). The pathogen is now well established in the Los Angeles basin and has forced the grape industry to move to northern California where the incidence of Pierce's disease is tolerable.

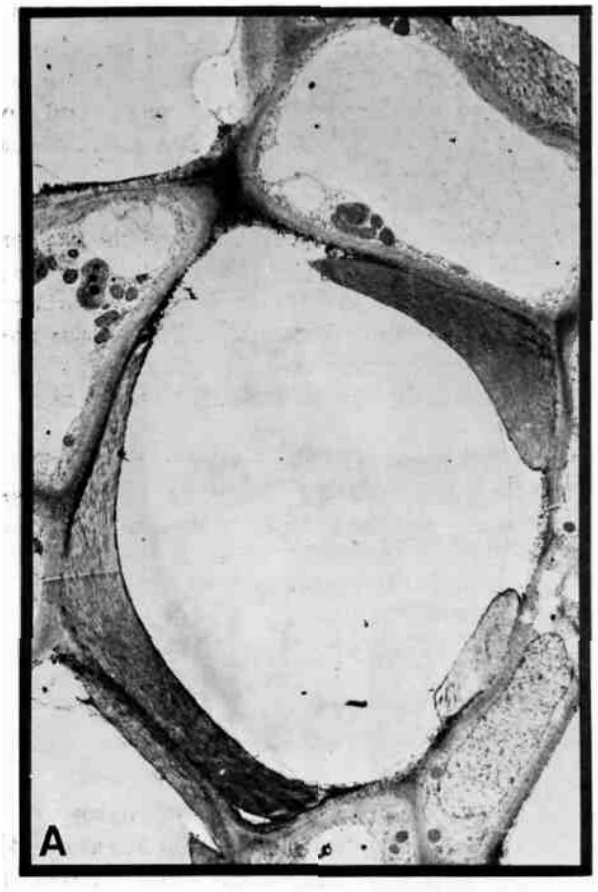

Fig. 1A. Xylem tissue of healthy grapevine.

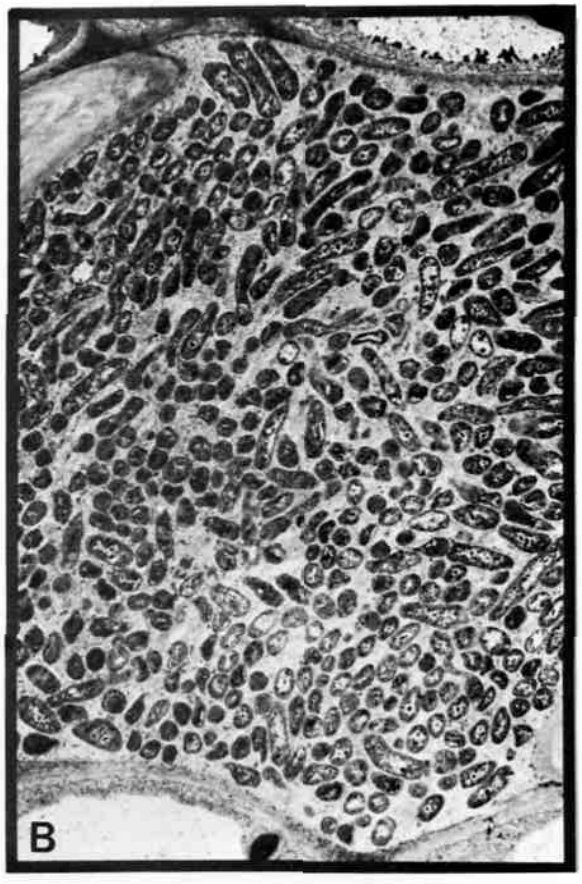

Fig. 1B. Xylem tissue of grapevine infected with Pierce's disease bacterium.

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ETIOLOGY. Soon after an unknown disease began to ravage the vines in southern California in the 1880s, the State Viticultural Commission appointed Ethelbert Dowlen to determine the cause of the so-called California vine disease (4). Dowlen believed the cause to be infectious and most likely fungal. He observed fungi on the necrotic leaves, however, he did not make isolations or identifications. In 1892, Newton B. Pierce (15) published a monograph on the California vine disease. Pierce believed that a bacterium was the causal agent because he observed bacteria in diseased tissue. However, he was not able to reproduce the symptoms of the disease with any of the bacteria that he isolated from affected grapes. By the 1930s a viral etiology was accepted because the causal agent could not be isolated in culture and the causal agent could be spread by grafting. In 1971, Hopkins and Mortensen (11) noted a suppression of symptoms after application of tetracycline antibiotics to the root zone of affected grapevines. This finding suggested a nonviral cause. In 1973, Hopkins and Mollenhauer (9) in Florida and Goheen, Nyland, and Lowe (5) in California simultaneously published electron micrographs that showed rickettsia-like bacteria in the xylem tissue of grape infected with Pierce's disease. Healthy vines did not harbor microorganisms. In 1974, Auger, Shalla and Kado (1) reported the isolation of a gram-positive bacterium from the leafhopper vector and the reproduction of symptoms of Pierce's disease on grapevines with this bacterium. However, proof of pathogenicity of this bacterium could not be repeated (13; D. L. Hopkins, unpublished information). In 1978, Davis, Purcell, and Thomson (2) isolated a gram-negative, catalase-positive, rod-shaped bacterium from grapevines affected with Pierce's disease. Koch's postulates were fulfilled by these workers and were also verified by others (D. L. Hopkins, unpublished information). The gram-negative bacterium has not yet been assigned to a taxonomic position.

SYMPTOMS. The bacterium that incites Pierce's disease is limited to the xylem tissue (fig. 1A and B). The major symptoms are similar to those of vascular wilt diseases. This similarity suggests that the disruption of the water-conducting system is the primary mode of disease development. Symptoms include decline of vigor (fig. 2), marginal necrosis of leaves (fig. 3), decreased production, smaller than usual grapes, and usually death of the plant.

The crown area is characteristically the last area of the plant to die of Pierce's disease (fig. 2). Since many pathogens and/or factors may cause similar symptoms, diagnosis of Pierce's disease requires that the bacteria be seen in the xylem tissue.

Fig. 2. Declining grapevine (foreground) infected with Pierce's disease bacterium compared to immune grapevines (background).

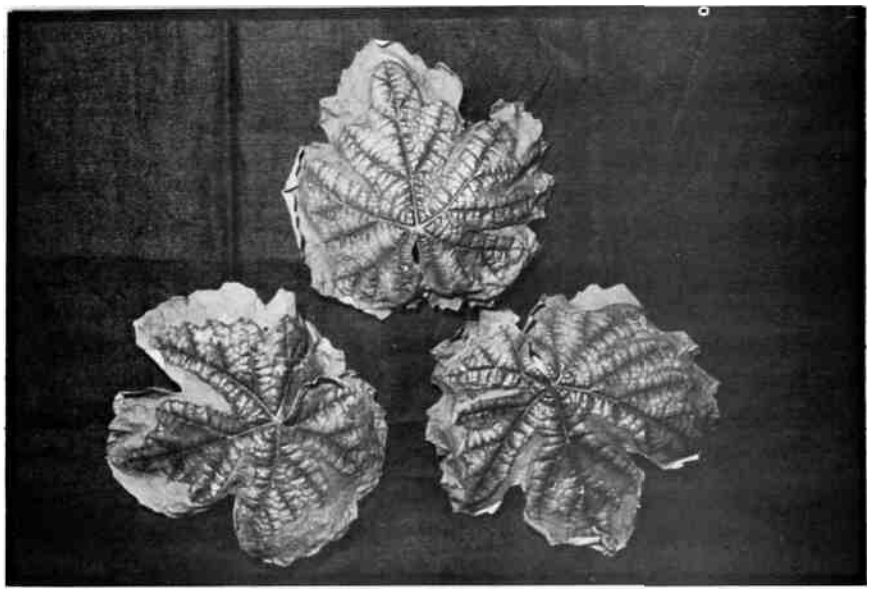

Fig. 3. Marginal necrosis of grape leaf from plant that is declining because of Pierce's disease. The youngest leaves quite frequently exhibit interveinal chlorosis.

DISSEMINATION. The disease-inciting bacterium is transmitted by sharpshooter leafhoppers in the subfamily Telligellinae. Houston, Esau, and Hewitt (12) in 1947 showed that the majority of leafhopper puncture wounds in grape stems terminated in the xylem tissue, and they demonstrated that Pierce's disease occurred only when the xylem tissue could be reached by the vector. Because of these observations, electron microscopists, working some 25 years hence, knew to look in the xylem for the causal agent. Whereas insect vectors can transmit the disease over short distances, the pathogen can be spread over much greater distances by man. The introduction of Pierce's disease into California in the 1880s was probably brought about by someone bringing infected grape wood from some area of the southeastern United States and grafting this wood onto southern California grapes (7).

HOST RANGE. The Pierce's disease bacterium has a wide host range. Hosts include 28 families of monocotyledonous and dicotyledonous plants (3, 6). Almond leaf scorch (14) and alfalfa dwarf (5) appear to be caused by the same agent that causes Pierce's disease. However, many of the hosts seem unaffected by the Pierce's disease agent (8).

CONTROL. The only control for Pierce's disease at present is genetically controlled resistance (8). For grapes to have a productive lifespan in the southeastern United States, resistant varieties are required. The grape industry in the Gulf Coastal Plain States is based upon native resistant Vitis spp. Muscadine grapes (V. rotundifolia Michx.), which are widely popular in the southeast, have a high degree of resistance to Pierce's disease, but all muscadines are not immune. In Florida, at least three muscadine cultivars ('Pride', 'Carlos', and 'Scuppernong') do not have enough tolerance to perform acceptably (10). Three Florida bunch grape cultivars ['Lake Emerald' (light green), 'Blue Lake' (blue), and 'Stover' (golden)] which were derived from Florida wild grapes are resistant, as well as a French hybrid, Roucaneuf (14).

Plants afflicted with Pierce's disease may be freed of the causal agent by hot water treatment (5). The entire plant is dipped in water at 45 C for 180 min, 50 C for 20 min, or 55 C for 10 min. This method eliminates any threat that Pierce's disease might be moved to new areas by diseased wood.

The broad-spectrum tetracycline antibiotics have been somewhat effective against the bacterium that causes Pierce's disease (8). Tetracyclines are more effective as protectants than as remitting agents. Information is needed on rates, frequency of applications, phytotoxicity, and cost-benefit analysis before adequate commercial control of Pierce's disease is feasible.

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